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Home environment and asthma in Kenyan schoolchildren: a case-control study

Nashila Mohamed, Lucy Ng'ang'a, Joseph Odhiambo, Janet Nyamwaya, Richard Menzies

Abstract

Background **– There is increasing evidence that environmental factors contribute to the development of asthma, so the relationship was studied between home environment factors and asthma among school children of varying socioeconomic backgrounds living in a developing country.**

Methods **– A case-control study was performed in participants of a prevalence survey which included 77 schoolchildren with asthma (defined by a history of wheeze, doctor diagnosis, or a decline in FEV_1 of $\geqslant 10\%$ at five or 10 minutes after exercise) and 77 age and gender matched controls. Subjects were selected from 402 school children aged 9–11 years attending five primary schools in the city of Nairobi who participated in a prevalence survey of asthma. Visits were made to the homes of cases and controls and visual inspection of the home environment was made using a checklist. A questionnaire regarding supplemental salt intake, parental occupation, cooking fuels, and health of all children in the family was administered by an interviewer.**

Results **– In multivariate analysis the following factors were associated with asthma: damage caused by dampness in the child's sleeping area (adjusted odds ratio (OR) 4·9; 95% confidence interval (CI) 2·0 to 11·7), air pollution in the home (OR 2·5; 95% CI 2·0 to 6·4), presence of rugs or carpets in child's bedroom (OR 3·6; 95% CI 1·5 to 8·5). Children with asthma reported a supplemental mean daily salt intake of 817 mg compared with 483 mg in controls.**

Conclusions **– Home environmental factors appear to be strongly associated with asthma in schoolchildren in a developing nation. These findings suggest a number of hypotheses for further studies.**

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Keywords: asthma, children, respiratory health, urbanisation, home environment.

Department of Epidemiology and Biostatistics
N Mohamed

Respiratory Epidemiology Unit
R Menzies

International Respiratory Diseases Research Unit of the IUAT-LD, McGill University, Montreal, Quebec, Canada H3A 1A3

Respiratory Diseases Research Centre, Kenyan Medical Research Institute, Nairobi, Kenya
L Ng'ang'a
J Odhiambo
J Nyamwaya

Reprint requests to:
Dr R Menzies.

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In 1987 the population of sub-Saharan Africa was estimated to be 350 million; most were less than 20 years of age and lived in households with low income.[1] Kenya has an annual population growth rate of 3·8%, the highest in the world.[2] Nairobi, the capital city, has experienced rapid growth and industrialisation over the last 20 years.[3] The effects of this urbanisation and industrialisation upon the respiratory health of children in Kenya have not been studied.

Over the past 20 years the prevalence and incidence of asthma has increased in many industrialised nations,[4] although the reported prevalence has ranged from 1·2% in Scandinavian countries[5] to 19% among children in New Zealand.[6] In developing countries the prevalence of childhood asthma ranges from 0·007% in Papua, New Guinea[7] and 3·3% in rural Tanzania[8] to 9·8% in urban Abidjan.[9] In a recent study among Nairobi schoolchildren the prevalence of asthma based on questionnaire responses was 11·4%, while 10·7% had exercised-induced bronchospasm.[10]

There is increasing evidence that environmental factors, as well as host factors, contribute to the clinical manifestations of asthma. Differences in the prevalence of asthma between industrialised and developing countries,[4] and urban–rural differences within developing countries,[11–13] suggest that environmental factors associated with urbanisation and industrialisation may play an important part. Putative environmental factors include exposures in the home environment, outdoor air pollution, and diet. We have conducted a study to investigate the possible association of these factors with indicators of asthma detected among participants of a prevalence survey recently completed among Nairobi schoolchildren.[10]

Methods

A case-control study among participants in a prevalence survey was conducted in the city of Nairobi which is situated in the highlands area of Kenya. Potential study subjects were children aged 9–11 years, attending grade 4 in public primary school in Nairobi. Schools were categorised on the basis of socioeconomic characteristics of the neighbourhoods in which they were located as slums, lower middle class, middle class, upper middle class, and city centre. Children attending city centre schools tended to be resident in all of these areas.

The parents or guardians of the children gave informed consent and completed an interview-administered asthma questionnaire (adapted from that developed by the International Union Against Tuberculosis and Lung Diseases). Lung function tests were carried out using a Vitalograph compact spirometer. Predicted values for forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were taken from prediction equations de-

veloped by Hsu[14] for Black Americans. Subjects were asked to run as far as possible for six minutes around a premeasured course and spirometric tests were performed five and 10 minutes after completion of exercise.[10]

The subjects for the case-control study were selected from the participants of the prevalence study. A case was defined by either (1) a history of asthma or symptoms of persistent or frequent wheeze reported on the questionnaire, or (2) a decline in FEV_1 at five or 10 minutes after exercise of at least 10% compared with baseline.[15-17] Controls, selected from the same school and class and matched for age and gender, did not have either of these criteria for asthma.

It was estimated that, in order to detect an odds ratio of 2 for the association of a home environmental factor and asthma with a specificity of 95% ($\alpha=0{\cdot}05$) and a sensitivity or power of 80% ($\beta=0{\cdot}2$), a sample size of 75 cases and an equal number of controls was needed.[18]

Within one week of participating in the prevalence study the homes of cases and controls were visited to inspect the house and administer a qestionnaire. Using a checklist, building material of the house, ventilation characteristics (number of windows and presence of chimney), type of bedding material used by the child, presence of rugs, carpets, sofas, or pets, type of cooking fuel, and visible air pollution within and outside the house were evaluated by one of the authors (NM).

The interviewer-administered questionnaire included items on the age, gender, and occupation of the employed members of the household, vaccination status, birth history, and mortality of all children in the family. Childhood mortality for the family was calculated as the total number of deaths in childhood divided by the total number of live births. Crowding index was calculated as the total number of occupants of a dwelling divided by the number of rooms. Supplemental salt intake of the case or control was estimated from questionnaire items on the use of added salt during meals and consumption of popular salt-containing snacks. The total weekly supplemental salt intake was calculated from the responses given, based on standard values of salt present in similar snack foods in North America.[19]

At the time of these home visits the investigator was blinded as to the status of the cases and controls. For logistic reasons blinding was possible only in the first three schools and not in the last two schools studied.

DATA ANALYSIS

All analyses were conducted using SAS program (SAS Institute, Carey, North Carolina, USA) except for logistic regression which was conducted using BMDP (BMDP Statistical Software, Los Angeles, California, USA). For dichotomous independent variables such as presence or absence of an environmental factor, odds ratios and 95% confidence intervals were calculated. For continuous variables such as supplemental salt intake *t* tests were used to test whether differences detected were statistically significant. Conditional logistic regression was used to calculate odds ratios and 95% confidence intervals adjusted for other risk factors and for potential confounding factors.[20]

Results

In the five schools studied between October 1990 and February 1991, 409 of 597 (69%) eligible schoolchildren participated. Of these participants, 77 (19%) in the prevalence survey[10] and an equal number of matched controls were identified. Thirty seven (48%) of the cases were diagnosed on the basis of exercise-induced bronchospasm, 20 (26%) by questionnaire, and 20 (26%) – or 5% of participants in the prevalence survey – by both means.[10] If the diagnostic criteria for case identification were changed to a decline in FEV_1 after exercise of at least 15%, 65 cases would have been identified (38·5% by exercise alone, 38·5% by questionnaire alone, and 23% by both criteria).

Before exercise baseline FEV_1 averaged 96·4% predicted among cases and 98·3% predicted among controls (NS), while FEV_1/FVC ratios averaged 86·6% and 89% respectively (NS). In the cases FEV_1 declined by a mean of 11·9% five minutes after exercise and 12·5% 10 minutes after exercise and by 2·1% and 2·6% respectively in the controls.

There were 13 childhood deaths and 327 live births among the families of cases compared with six childhood deaths and 351 births among controls for overall crude childhood mortality rates of 39·8 and 16·6 per 1000 live births respectively (table 1). The national average for Kenya was 111 per 1000 live births in 1990.[2] Of the participants, 98% were fully immunised; this is much higher than the national average of 75%,[3] perhaps attributable to the high priority given to immunisation programmes in the city of Nairobi.[3] The crowding index, vaccination coverage, and parental status were not significantly different between cases and controls.

There were significant differences in types of housing, sanitary conditions, crowding index, and childhood mortality between those residing in the different neighbourhoods. Of the children living in the slums, 88% lived in mud or semi-completed houses, 96% used a pit latrine, and crude childhood mortality was 56 per 1000. Of those residing in the lower middle class areas, 5% lived in mud or semi-complete homes, 10% used pit latrines, and crude childhood mortality was 19 per 1000. Of the children living in the middle and upper-middle class neighbourhoods, 89% lived in brick homes and none in mud or semi-complete homes, 83% had flush toilets, and crude childhood mortality was 12 per 1000. The average number of people per room in each household was three times higher in the slums than in the middle class areas. The number of people sharing the child's bedroom in the slum and lower-middle class areas was double that of middle class areas. Matching cases and controls by school did not always ensure matching by neighbourhoods as some students resident in more affluent neighbourhoods attended schools in the slum

Table 1 Characteristics of study population

	Cases	Controls	p*
No.	77	77	
Mean age (years)	10·2	10·2	
M:F	36:41	36:41	
Type of school (n)			
City school	15	15	
Slum school	15	15	
Middle class	14	14	
Upper middle class	15	15	
Low middle class	18	18	
Mean crowding index (no. of people/no. of rooms)	1·9	2·0	NS
Vaccination status (no. complete)	75	76	NS
Childhood mortality (per 1000 live births)	39·8	16·6	<0·05
No. people sharing child's bedroom	3·1	3·6	<0·05
Parental status			
Monoparental	11	19	NS
Biparental	66	58	

* Paired t test or χ^2. Subjects matched on first three variables.

Table 2 Potential exposure of cases and controls

	Cases	Controls	p*
Cooking fuels:			
Wood and charcoal	17 (22%)	8 (10%)	NS
Kerosene	20 (26%)	23 (29%)	
Gas	31 (40%)	32 (41%)	
Electricity	9 (12%)	14 (18%)	
Housing type:			
Dampness in bedroom and mud floors	4 (5%)	5 (7%)	NS
Dampness in bedroom and no mud floors	34 (44%)	9 (12%)	
No dampness in bedroom and mud floors	0 (0%)	3 (4%)	
No dampness in bedroom and no mud floors	39 (51%)	60 (78%)	
Extra salt intake	817 mg	483 mg	<0·0005

* Paired t test or χ^2.

Table 3 Unadjusted odds ratio of home environmental factors and asthma

Home environment factor	*Crude odds ratio*	*95% Confidence interval*
Damp damage in house	2·1	1·1 to 4·1
Damp damage in child's bedroom	4·4	2·1 to 9·1
Air pollution in house	3·0	1·4 to 6·3
Air pollution around house	2·1	1·1 to 4·1
Rugs, carpets or furniture in child's bedroom	2·9	1·4 to 6·3
Smoking by a member of the household	1·6	0·8 to 3·2
Presence of mud walls	1·1	0·4 to 2·8
Presence of mud floors	0·6	0·2 to 1·9

areas because of the better reputation of a particular school.

As shown in tables 2 and 3, asthma was associated with the presence of visible mould or damage due to dampness, particularly if this occurred in the child's sleeping area. Houses with mud walls were five times more likely to have damage resulting from dampness than homes with wooden or cement walls (OR 5·0; 95% CI 1·9 to 13·3). Similarly, houses with mud floors were eight times more likely to have damage caused by dampness than houses with cement or wooden floors (OR 7·8; 95% CI 2·1 to 30·0). However, despite this association, the presence of mud floors was not associated with asthma.

At the time of the home visits the observer was blinded to the status of cases and controls among participants from the first three schools, but not for the latter two schools. To detect any potential observer bias, odds ratios of asthma with damp damage and indoor and outdoor pollution were calculated separately for the first three schools (blinded) and compared with those of the last two schools studied (not blinded). No significant differences were seen – for example, the odds of damage due to dampness being present in homes of cases compared with controls was 6·8 (95% CI 2·6 to 17·7) in the schools that were blinded and 4·6 (95% CI 1·5 to 15·1) in schools that were not.

Observer assessed indoor air pollution was also associated with asthma. Visible indoor air pollution was five times more likely if kerosene was used as a primary cooking fuel and three times more likely if charcoal was used than in homes using neither. Despite the association of indoor air pollution with cooking fuels, cooking fuels were not associated with asthma and the presence of indoor air pollution was not related to the time of the home visit. Homes considered to have significant indoor air pollution had an average of 4·3 windows compared with an average of 7·1 windows in homes without indoor air pollution ($p<0·005$). Indoor air pollution was also associated with visible air pollution outside the house (crude OR 6·2, 95% CI 2·9 to 13·8).

As shown in table 3, visible air pollution outside the house was significantly associated with asthma. Households in the slum and lower-middle class neighbourhoods were seven times more likely to have outdoor air pollution than those in the middle and upper-middle class neighbourhoods. The presence of factories in the vicinity of the home was assessed visually by standing outside the home and recording if factories could be seen. If seen, air pollution outside the home was 16 times more likely to be present.

Three of the mothers, 37 of the fathers, and 13 other members of the households smoked so that at least one adult smoked in 48 of the homes. Cigarette smoking by any family member in the home was somewhat, but not significantly, associated with asthma (OR 1·6; 95% CI 0·8 to 3·2). When analysed separately there was no significant association between asthma in the child and cigarette smoking of the mother, father, or other members of the household. Smoking in the home was not significantly associated with indoor air pollution (OR 0·8; 95% CI 0·4 to 1·7).

As shown in table 4, asthma was significantly associated with the presence of damage caused by dampness, the presence of rugs, carpets and furniture in the child's sleeping area, indoor air pollution, and supplemental salt intake when conditional logistic regression was used to obtain estimates of assocation adjusted for other risk factors. After adjustment for these four factors, outdoor air pollution, the presence of mud walls or floors, and passive smoking were not significantly associated with asthma. A final model with four significant factors and the

Table 4 Adjusted odds ratio of home environmental and dietary factors and asthma

*Variable**	*Odds ratio*	*95% Confidence interval*
Damp damage in child's bedroom	4·9	2·0 to 11·7
Furniture, rugs and carpets in child's bedroom	3·6	1·5 to 8·5
Air pollution in the house	2·5	2·0 to 6·4
Extra salt intake of the child†	1·6	1·1 to 2·4

* Air pollution outside home, passive smoking, mud floors, and mud wall were not significantly associated in multivariate analysis.
† Odds ratio is for (mean + 1 SD) compared with (mean) daily salt intake.

interaction term of damage caused by dampness with the presence of rugs or carpets in the child's sleeping area was tried. In this model the interaction term was not significantly associated with asthma.

Discussion

Among Nairobi schoolchildren aged 9–11 of varying socioeconomic backgrounds, 77 were identified as having asthma on the basis of exercise-induced bronchospasm and/or questionnaire responses. These were matched to 77 children of the same school, class, age, and gender without either of these markers of asthma. Important associations were found between asthma and several home environmental factors as well as supplemental salt intake.

Potential limitations of the study include concerns regarding the representativeness of the study population, and the lack of objective measures such as fungal or house dust mite antigens or indoor air pollutants. About 68% of all schoolchildren in Nairobi attend public schools,[3] of whom 69% participated; thus, 47% of the total estimated eligible population participated in the prevalence survey. The prevalence of asthma may have been overestimated or underestimated if the prevalence of asthma among the children not attending school, or those not participating in the study, differed from the participants. However, the present study of the association of home environmental factors with asthma should not have been biased because both cases and controls were drawn from the same population, and were therefore subject to the same selection factors. The participation rate of 100% in the case-control study should have limited any additional potential selection bias. The selection factors in the prevalence survey may, however, limit the generalisation of the results.

The selection of controls from the same schools as cases may have limited the ability to detect effects of socioeconomic status because most children in the same schools were from similar socioeconomic backgrounds. However, there was sufficient variation in many home environmental factors (such as mud walls or floors) for these effects to be detected.

The definition of asthma used in this study to identify cases may have been different from that used in other studies. This may be important because factors associated with exercise-induced bronchospasm may be different from those associated with asthma diagnosed by questionnaires.[21] The present study did not attempt to investigate the association of risk factors within subsets of asthma defined by different criteria.

Measurement of some of the environmental risk factors such as damage caused by dampness or indoor or outdoor air pollution was subjective, and so potentially subject to observer bias. This was controlled by using a standardised format to collect data in the homes of cases and controls, and by establishing a priori criteria for ascertaining all exposure variables. To further reduce potential observer bias, the researcher was blinded to the status of the subject during visits to homes of participants from the first three schools. Where blinding was not possible, analyses were consistent with absence of bias.

Salt intake was strongly associated with asthma in this study, although this information was obtained solely by diet recall and estimated only supplemental salt intake. This information was collected in a standardised way and these findings are consistent with other studies which show an association between salt intake and bronchial hyperreactivity.[22,23]

In about half the world's households, particularly those in developing countries, biofuels such as wood, crop residues, and animal dung are used for cooking daily, usually without chimneys and with poor ventilation leading to high levels of smoke indoors.[24] Acute respiratory infection in children in six developing countries was significantly associated with levels of indoor air pollution.[25] Respiratory health symptoms, including those of asthma among children and adults in Britain and North America, have not been consistently associated with domestic air pollution.[26,27] In the present study increased indoor air pollution was associated with asthma in bivariate and multivariate analyses. The stronger and more consistent associations between respiratory health and indoor air pollution in studies in developing countries may be a reflection of the much greater intensity of exposure in homes in these countries. In a survey of homes in Stockholm, house dust mite antigens and resulting allergic reactions were more prevalent in children in homes with poor ventilation,[28] suggesting another mechanism for the relations found in this study.

Another important source of indoor air pollution is passive smoking. In a number of studies parental smoking has been associated with persistent wheeze and cough among children.[26,29] The lack of effect in this study may have been because of the relatively small sample size or because, in developing countries, many of those who smoke actually smoke only a few cigarettes per day.

Respiratory symptoms including chronic cough and wheeze have been associated with dampness and/or damage caused by dampness in homes in previous studies among children[21,26,30] and adults.[31] Airborne fungal concentrations are higher in damp houses than in dry houses.[30] The presence of visible indoor mould, especially penicillium, was significantly associated with asthma among those aged 15–60 in Cardiff.[32] This evidence has led many

to believe that there may be a causal relation between exposure to fungal antigens at home and the development of asthma.[21 31] However, there is also abundant evidence that exposure to the house dust mite in the home may lead to the development of asthma.[28 33] Since both the house dust mite and fungi thrive in damp conditions,[33] either or both may be important in the pathogenesis of asthma. In this study asthma was associated with dampness, and independently with the presence of rugs, carpets, or bedding which may act as reservoirs for both house dust mites and fungi.[28 33] To test the biologically plausible hypothesis that dampness may interact with the presence of rugs and other furnishings to amplify exposure to fungi or house dust mite, we tested the interaction term of these two factors in the regression model. No significant association was seen between the presence of asthma and this interaction term, either because of the absence of any biological interaction or because of the small sample size of the study.

The strong association of mud walls and mud floors with damage caused by dampness was expected. However, while mud walls were also significantly associated with asthma in bivariate analysis, children in homes with mud floors were less likely to have asthma. It is interesting to speculate if asthma was less likely in these homes because house dust mites do not survive in soil, or because of differences in the fungal species present in the soil. Since we did not measure levels of house dust mite or fungal antigens, this finding needs to be tested in further studies.

The prevalence of asthma in many parts of Africa seems to have increased considerably,[4 8 10–13] a phenomenon which may be explained by urbanisation and the accompanying changes in life style.[11–13] In this study in schoolchildren in Nairobi factors in the home environment were strongly associated with asthma. These results generate hypotheses for future studies on the effect of fungal and house dust mite antigens and indoor air pollution on the respiratory health of children in developing countries.

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